REMARKS

Reconsideration of the application as amended is requested.

New claims 12 and 13 have been added which are directed to primer extension methods using specific extension primers. Support for new claims 12 and 13 is found throughout the application, appearing specifically at paragraphs [030], [032], [035], and [040] and in Figure 1E. No new matter has been added by virtue of the amendments to the claims.

Rejections pursuant to 35 U.S.C. § 103

Claims 1-5 stand rejected as being unpatentable over Rutledge et al. in view of Hattori et al. (1995) and Sathasivan et al. The Examiner takes the position that Rutledge et al. teaches the sequences of the *B. napus* AHAS1 and AHAS3 genes, isolation of DNA from leaf nuclei, that herbicide resistance of *B. napus* mutants results from two unlinked alleles, and that the effect of combining the alleles in a hybrid line is additive. The Examiner admits that Rutledge et al. is deficient in a teaching of the nature of the mutations in AHAS 1 (PM1) and AHAS 3 (PM2) that confer resistance to imidazolinones. The Examiner opines that the deficiencies of the primary reference are overcome by the disclosure of the PM2 mutation in Hattori et al. in combination with the teaching of the G to A point mutation at nucleotide 1958 of the ALS gene from the homozygous *Arabidopsis thaliana* mutant line GH90. This rejection is respectfully traversed.

Applicants wish to clarify the Office Action's reference to page 39, right column, last paragraph of Rutledge et al., which did state that "[t]his study provides the basic information essential for the analysis of transgenic *Brassica* crops or mutants with resistance to herbicides that act on AHAS". However, in the next sentence the reference also states that. . . "[t]he extent of genetic complexity within the AHAS gene family was much greater than anticipated." In fact, at the beginning of the Discussion section on page 39, Rutledge et al. state that . . . "[t]he complexity of the AHAS multigene family was far more extensive among the *Brassica* species than reported for *A. thaliana* or *N. tabacum*." *(citations omitted)*.

The Examiner has acknowledged that Rutledge et al. is entirely deficient in any disclosure of the PM1 and PM2 mutations. Rutledge et al. does not teach or suggest the specific molecular modifications necessary to achieve the PM1 and PM2 mutations of the native *B. napus* AHAS1 and AHAS2 genes. *See, In re Deuel*, 34 USPQ2d 1210, 1214 (Fed. Cir. 1995). Rutledge et al. therefore provides no more than a general prediction about the *B. napus* alleles that conferred resistance to sulfonylureas and imidazolinones.

Although Hattori et al. (1995) discloses a mutation from a herbicide-resistant *B. napus* callus suspension culture that corresponds to the PM2 mutation, the secondary reference is devoid of any teaching of the PM1 mutation. Moreover, the Hattori et al. (1995) is also devoid of any teaching of the relationship between the disclosed mutation and commercially relevant levels of imidazolinone tolerance in a *Brassica* plant.

Moreover, Hattori et al. (1995) discloses that many mutations in the AHAS gene may result in herbicide tolerance. For example, at page 420, left column, Hattori et al. (1995) states that . . ."[it] is known that AHAS may accumulate several mutations. . .each of which may contribute independently to the phenotype" *(citations omitted)*. Further at page 424, left column, Hattori et al. (1995) states that:

> [a] total of ten separate sites within AHAS are known to be involved in sulfonylurea binding, but the patterns of cross-resistance to other herbicides or amino acids have not been reported for most of them. *(citations omitted)*

Hattori et al. (1995) does not teach or suggest which of these ten sites could correspond to the PM1 mutation.

Hattori et al. (1995) states in the concluding paragraph of page 424 that:

> [u]nfortunately, the instability and small amounts of AHAS in plants such as rapeseed, Arabidopsis and tobacco have impeded attempts to purify the enzyme in its native form and thus define its structure and regulatory properties. *(citations omitted)*

Thus Hattori et al. (1995) provides evidence of the difficulties that existed in defining the molecular basis of the PM1 mutation.

Sathasivan et al. fails to cure the deficiencies of Rutledge et al. and Hattori et al. as applied to the PM1 mutation. Sathasivan et al. actually discloses several mutations that one of skill in the art could believe to be analogous to the *Brassica* PM1 mutation (*see*, Table I). Given the complexity of the *Brassica* AHAS gene family disclosed in

Rutledge et al., the number of different possible herbicide resistance mutations in AHAS and the difficulty in defining the structure of the AHAS enzyme as disclosed in Hattori et al. (1995), the identity of the PM1 mutation would not have been "evident" to one of skill in light of Sathasivan et al.

None of the references provides direction as to which of the many possible AHAS mutations could be PM1, nor do the references indicate which of many possible parameters would have been critical to determine the identity of PM1. As stated by the Federal Circuit, "'Obvious to try' has long been held not to constitute obviousness." *In re Deuel*, 34 USPQ2d at 1216, citing *In re O'Farrell*, 7 USPQ2d 1673, 1681 (Fed. Cir. 1988). Withdrawal of the rejection under § 103 over Rutledge et al. in view of Hattori et al. (1995) and Sathasivan et al. is therefore respectfully requested.

Claims 1-5 stand rejected as being unpatentable over Rutledge et al. in view of Hattori et al. (1995) and Hattori et al. (1992). The Examiner reiterates his characterization of Rutledge et al. and Hattori et al. (1995) and applies Hattori et al. (1992), which discloses the *A. thaliana imr1* mutation, in a manner similar to the application of Sathasivan et al. in the previous rejection. This rejection is respectfully traversed.

Applicants' comments above regarding the deficiencies of Rutledge et al. and Hattori et al. (1995) as applied to the PM1 mutation are incorporated by reference into this traversal.

Similar to Sathasivan et al., Hattori et al. (1992) fails to specify the identity of the PM1 mutation, and in fact states that . . ."[i]t is likely that mutations at other sites will be found which yield imidazolinone resistance once more mutant AHAS genes are characterized."[1]

None of the references provide direction as to which of the many possible AHAS mutations could be PM1, nor do the references indicate which of many possible parameters would have been critical to determine the identity of PM1. The Federal Circuit's prohibition against applying the "obvious to try" standard is equally applicable to the instant rejection. *In re Deuel*, 34 USPQ2d at 1216 . Withdrawal of the rejection

[1] Page 172, right column.

under § 103 over Rutledge et al. in view of Hattori et al. (1995) and Hattori et al. (1992) is therefore respectfully requested.

Double Patenting

Claims 1-5 stand rejected under the judicially created doctrine of obviousness-type double patenting over USSN 10/695,546. Applicants are willing to provide a terminal disclaimer over USSN 10/695,546 upon an indication of allowability of the present claims.

In light of the amendments and arguments set forth above, Applicants submit that all of the rejections contained in the Office Action dated August 28, 2006 have been overcome, and the application is in condition for allowance. Should the Examiner wish to discuss the application further, he is invited to telephone the undersigned. If any additional fees are due with respect to this submission, authorization is hereby given to charge such fees, or to credit any overpayment, to Deposit Account No. 02-1197.

Respectfully submitted,

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